British Heart Journal, 1973, **35**, 858–867.

Proceedings of the British Cardiac Society

THE FIFTY-SECOND ANNUAL GENERAL MEETING of the British Cardiac Society was held at the University of Glasgow on Thursday, 12 April 1973. The President, JOHN GOODWIN, took the Chair at 9.00 a.m. during Private Business. At the Scientific Session which followed the Chair was taken by OLAV KERR.

Private Business

1 The President reported with deep regret the death of Heim de Balsac, a Corresponding Member who was well known to the Society and who had worked with Ian Hill in Dundee for a time; Ben Lassers, a young member of the Society who died in a tragic accident recently; and finally Dickinson Richards who though not a member of the Society deserved mention because of his eminence in the field of cardiology, and the fact that he had shared the Nobel Prize for Medicine with Cournand and Forssmann for the introduction and development of cardiac catheterization.

2 The Minutes of the Autumn Meeting would be published shortly in the *British Heart Journal*.[1]

3 The Treasurer presented the audited accounts for the year ended 31 December 1972. A few copies of the accounts were available for members to see if they wished, and he would be pleased to explain any points that were not clear. He mentioned in particular the excess of expenditure over income of £80, and the fact that the investment income of £572 had been used to meet the running expenses of the Society in 1972. Council were recommending increasing the ordinary membership to 400, which would increase the Society's income; but with the steadily rising costs Council recommended, and he now formally proposed, that the annual subscription should be raised to £15. This would include the subscription to one of the Journals, as now, but would also cover the cost of coffee and tea at the Society's scientific meetings. This was carried, and the audited accounts were adopted and signed.

Investments at the end of the year had a book value of £20,780 against a cost of £13,717, but the value would have fallen off slightly in line with the decline in the stock market. Oram had kindly agreed to join the small team advising about investments and it was hoped to arrange a meeting with the new broker, Mr. Minford, very soon.

It seemed almost certain that the Society as a professional body would not be involved in Value Added Tax, but final confirmation was awaited from the Inland Revenue.

The Treasurer reported that the auditors had been extremely helpful in streamlining the book-keeping and suggesting methods of simplifying the work and so saving the Society's money. Both he and the Executive Secretary had been worried for some time about the enormous amount of time necessary to run the present system of meal cards, and, in spite of the most careful checking, the loss of money. Members might not be aware that the attitude of caterers had hardened considerably, they demanded exact numbers well in advance, and there was no concession for meals not consumed. Some members had in the past ordered meals, particularly dinners, and had not paid for them, and often this money could not be recovered in spite of protracted correspondence; the resulting loss to the Society could be as much as £100 on a dinner. Members would no doubt agree that this was not a satisfactory standard of conduct, and the auditors strongly advised a system of payment in advance.

In future, therefore, meals must be paid for in advance by cheque, and the reservations and cheques must be received by the deadline which is announced. Meals would not be available for members who had not paid on time, and there could be no exceptions. The need for the change was regretted, but it had really been forced upon the Officers by the increasing size and complexity of the Society, the rising costs, and by the inflexible attitude of caterers.

There would probably always be a few members who had ordered and paid for meals in advance and who at the last moment could not attend. If such members would advise the Executive Secretary immediately she would endeavour to sell the meals to any latecomers. A latecomer who had not reserved meals could ask at the desk on the day for any meals he wanted, and hand in his cheque for the appropriate amount. He would then either receive the meal cards if there were cancellations, or his cheque would be returned to him.

The Treasurer stressed that for the efficient running of the meetings at a reasonable cost it was essential that members should co-operate by reading the notices carefully and keeping to the deadlines.

4 Amendments to Rules

a) Council recommended that the number of Ordinary Members should be increased to 400, and that Rule 7 should be amended to read:

> 'Ordinary Members shall not exceed 400 in number, including those who may be elected for their interest in Cardiovascular Surgery (Surgical Members).'

Gibson felt that there was no need for a limit on the numbers since all proposed members would be approved by Council. This was supported by Balcon. Whitaker felt that the Society should be restricted in number and

[1] Published in May 1973, **35**, 548.

should remain at 300 Ordinary Members or possibly even be reduced.

Arnott requested a referendum among the members of the Society on the question of restricting the number of members, and suggested that a voting slip should be sent out with the papers for the 1973 Autumn Meeting.

The amendment to Rule 7 was carried, but Council would consider the suggestion of a referendum at the next meeting.

b) The following amendment to Rule 8 was proposed and agreed:

'Every Ordinary Member is required to attend at least one of any four consecutive Meetings of the Society. Failure to do so, without an explanation that is satisfactory to the Council, shall automatically terminate his membership of the Society.'

c) The following amendment to Rule 13 was proposed and agreed:

'In addition to those specified in Rule 7, Overseas Members may be elected from British-trained Cardiologists employed abroad who would otherwise be eligible for Ordinary Membership.'

d) The following amendment to Rule 22 was proposed and agreed:

'The subscription shall be fixed by the Council and shall become payable on the 1st day of June. For Ordinary Members it is £15·00, and for Overseas Members £5·25. This includes the subscription either to the *British Heart Journal* or to *Cardiovascular Research*, one of which is compulsory. Failure to pay the subscription due within two years shall be considered equivalent to resignation.'

5 Gray and Pentecost were elected to the Council to replace Counihan and Daley who had completed their term of office. Under Rule 21 Barber was co-opted to represent Ireland.

6 The following resignations on retirement were accepted:

E. F. Gartrell (from Corresponding Membership) and R. A. Jones, J. W. Litchfield, and R. Walmsley,

and also the resignation from Ordinary Membership of P. J. Molloy

who was going to New Zealand.

7 The following were elected *Extra-Ordinary Members* of the Society:

P. R. Allison, E. Wyn Jones, W. Arthur Mackey, P. H. O'Donovan, C. Papp, W. Phillips, D. A. Robertson, A. Schott, G. Simon, and O. S. Tubbs.

8 The following were elected *Corresponding Members* of the Society:

H. Denolin	Brussels, Belgium.
H. O. Wong	Kuala Lumpur, Malaysia.
Charles Toh	Singapore.
J. Barlow	Johannesburg, South Africa.
A. Holmgren	Stockholm, Sweden.
E. Varnauskas	Goteborg, Sweden.
R. Gorlin	Boston, U.S.A.
Richard Ross	Baltimore, U.S.A.

9 The following *Ordinary Members* were elected:

A. A. Jennifer Adgey	Belfast
Terence English (SM)	London
John Fowler	Basingstoke
Peter Michael Stephen Gillam	Salisbury
Peter Hewlett Kidner	London
Keyvan Moghissi	Hull
Winifred Gwen Naylor	London
John Parker (SM)	London
Maurice Nellen (from Corresponding Member)	London
Brian N. C. Prichard	London
R. B. Pridie	London
Geoffrey Arthur Rose	London
Jean Shackleton	Liverpool
Eric Dale Silove	London
Geoffrey H. Smith (SM)	Sheffield
Jaroslav Stark (SM)	London
Martin Geoffrey Thorne	Torquay
Geoffrey Herbert Watson	Manchester
William Gilbert Williams (SM)	Coventry/Birmingham
John Stephen Wright	Liverpool

10 The following *Overseas Members* were elected:

L. Gonzalez-Lavin, Detroit, U.S.A.
P. J. Molloy, Dunedin, New Zealand
} transferred from Ordinary Membership

Arni Kristinsson, Reykjavik, Iceland.

11 The Secretary confirmed that the Autumn Meeting 1973 would be held on 13 and 14 December at the Royal College of Physicians, London.

The suggestion had been made that at the 2-day meetings the first day should start at 10.00 a.m. and be devoted to Scientific Sessions, and the second day should start at 9.00 a.m. with the Private Business and continue with the remaining Scientific Sessions. This was agreed.

The Secretary announced that Council had carefully considered a number of suggestions for the 1973 Thomas Lewis Lecturer and had unanimously chosen Alf Holmgren of the Karolinska Hospital, Stockholm, who had accepted.

12 Dewar confirmed the invitation to hold the 1974 Annual General Meeting in Newcastle-upon-Tyne on 18 April, and stated that arrangements were well in hand.

13 The Secretary reported that the Swedish Society had confirmed their invitation to the British Cardiac Society to hold a joint meeting with them in Stockholm in September 1975, and Lars Mogensen had been appointed to make the arrangements in Stockholm. Quotations were being obtained for group travel and accom-

modation. It was hoped that a high proportion of Members would attend the meeting, which should be excellent in every way; naturally the larger the group the cheaper it would be. Further details of probable costs would be sent to members as soon as they were available.

14 Liaison Committee between British Cardiac Society, Cardiology Committee of the Royal College of Physicians of London, and the Department of Health and Social Security.

The President reported that the first meeting of this Committee, to be known as the Cardiovascular Liaison Committee, was held on 6 April 1973 with himself in the Chair. It was resolved that the Committee would act as a clearing house for information, a channel of communication between informed cardiological opinion and the Department of Health and Social Security, and a means of collecting important information. It would be able to define areas of need in many aspects of cardiovascular work, suggest new approaches, and act as a 'think tank'. It would not be an executive committee and would impose no sanctions nor cut across any established lines of authority or communication. It would maintain a link with the Joint Consultants Committee.

Subjects which would initially be considered by the Committee were – career structure; manpower and mobility of consultants; co-ordination of clinical trials; rationalization of purchase and development of apparatus; promotion and development of growing points; and technician training.

There would be up to six meetings a year. At the next meeting informal papers would be written on consultant mobility and co-ordination of clinical trials, respectively. Members of the British Cardiac Society were asked to send any suggestions, problems, or questions to Goodwin who had been greatly encouraged by the enthusiasm and informed co-operation of the members of the Committee from the Department of Health and Social Security.

15 Specialist Advisory Committee on Cardiovascular Disease to the Joint Committee on Higher Medical Training.

The President reported that a meeting of the Committee was due the following day as it had been impossible to arrange one for the day preceding the Cardiac Society's meeting. Arrangements for visiting centres for accreditation for training were in the process of development, a further meeting of the Joint Committee on Higher Medical Training would shortly be held and a further report would be made to the Society at the next meeting.

16 Society of Cardiological Technicians.

Towers had reported to the Society of Cardiological Technicians Ltd., at its annual meeting and in a letter to the Secretary of its Education Committee, the view of the Cardiac Society that the salaries of higher grade cardiological technicians would probably be linked in future to possession of the O.N.C. or H.N.C. It therefore behoved every technician to avail himself or herself of the academic training now available at various centres throughout the country. Towers had passed on to the Editor of the technicians journal and to the Secretary of their Education Committee the list of training centres prepared by Davison with the strong suggestion from the Cardiac Society that this list should be included in a future number of the technicians journal.

A number of members reported that no satisfactory training programme was available for cardiac technicians in their own areas. Block release courses held in London were not a complete or satisfactory answer. Many Departments were seriously understaffed and simply could not maintain their routine work and allow their technicians to go off on block release. In addition a broadly based programme in physiological measurement which might be ideal for technicians working in multidisciplinary teaching hospitals was a good deal more than the minimum required for the specialist cardiological technician working in a cardiac unit.

17 Working parties

The President reported that the Working Parties on Coronary Care and Rehabilitation, respectively, had been formed in conjunction with the Cardiology Committee of the Royal College of Physicians of London, with representatives from the Royal College of General Practitioners and observers from the Department of Health and Social Security.

a) *Coronary care*

The Chairman, Lawson McDonald, reported that the Committee was due to hold its first meeting shortly to discuss the terms of reference and appropriate courses of action.

b) *Rehabilitation*

The Chairman, Semple, reported on its activities, mentioning the two main aspects which were – (1) the problems of social and economic importance in rehabilitation, and (2) the physiological effects of effort on the cardiovascular system.

The Working Party was collecting information on the magnitude of the problems involved and subcommittees had been formed to discuss the two main issues mentioned.

c) *Prevention of infective endocarditis*

Hugh Fleming reported that he was gathering information on regimens of chemoprophylaxis for infective endocarditis and would be grateful if members could send him a note of their current practice or any schedules they might have.

d) *Suggested Working Party on the Primary Prevention of Cardiovascular Diseases*

The President reported that he had been approached by several members of the Society regarding the formation of this new Working Party, and that Council had agreed to this in principle. Oliver suggested that the terms of reference would need to be very clearly defined in order to avoid repetition and confusion with other bodies and committees dealing with the same problems. The President suggested that informal discussions should be held when information was forthcoming from the other bodies

which were shortly due to report on the problems. He suggested that the matter should be discussed again at the next meeting of the Society.

e) *Cardiac Muscle Research Group*
The President said that this group had already been formed with Peter Harris as Chairman, and that its relation to the Society with special regard to mutual membership and the presentation of scientific material to the Society was under active discussion. The Group had already arranged a meeting to take place on the day before the next Cardiac Society Meeting. Unfortunately, as Peter Harris was not able to be present today, further discussion would need to be postponed and a subsequent report would be made to the Society at its next meeting.

The President reported that the Royal College of Physicians had indicated their willingness to help towards the expenses of the two Working Parties with which they were connected. The Society's funds for expenses could therefore be held in reserve.

18 Young Research Workers' Award

The Secretary reported that he had formed the small sub-committee as requested, consisting of himself, John Hamer, and Derek Gibson. They were now drawing up draft Rules and a draft Notice to announce the inauguration of the Award, for submission to Council and the Society.

19 Correspondence

a) *VII World Congress of Cardiology, Buenos Aires, 1974*
The Secretary reported that several travel agents were preparing package deals, and at the moment it looked as though the cost would probably be over £400.

b) *Letter from Geoffrey Wade*
Wade invited the Society to hold the 1975 Annual General Meeting in Manchester, and the invitation was accepted with pleasure.

20 Any other business

Echocardiography
Hollman reported that it was proposed to hold a half-day Seminar on the present position of echocardiography in London during October.

The Society dined in the Hunter Hall of the University of Glasgow with the President in the Chair. Guests included Professor Eric Cruickshank, Postgraduate Dean of the Faculty of Medicine of Glasgow University, Professor Richard Gorlin and his wife, and Professor Bricaud from Bordeaux. The President thanked the Officers of the Society and the Executive Secretary for their help during the year and the Chairman, Olav Kerr, John McGuinness the Local Secretary, and all the Glasgow Members for their hospitality and for what had proved an extremely happy meeting. He also outlined the objectives and activities of the Society, with special reference to the importance of active original work and of representing British Cardiology in all its important aspects. The President congratulated Shirley Smith on his long and valuable service as Editor of the *British Heart Journal*.

Olav Kerr welcomed the Society to Glasgow, expressed his pleasure at having senior members of the Society at the Dinner, and gave some reminiscences of his former chiefs.

At the beginning of the Scientific Session the Chairman announced that he and the President had sent a joint telegram on behalf of the Society to Sir John Parkinson to reach him on the day of the meeting.

Glucose and insulin in ischaemic heart disease

S. H. Taylor, P. A. Majid, and B. Sharma (both introduced)

The anaerobic metabolism of glucose is the main energy source for ischaemic myocardium. Insulin is essential for the full myocardial utilization of glucose particularly during ischaemia. Insulin secretion is suppressed in heart failure. For these reasons the effects of glucose and insulin on left ventricular performance were studied in patients with ischaemic heart disease.

Six male patients in severe heart failure from ischaemic heart disease and 6 patients with exercise-induced angina pectoris were studied at rest and during exercise by conventional catheter techniques. Control observations were followed by 1·5 g/kg oral glucose and the intravenous infusion of 0·5 g/kg glucose, 1·5 I.U. soluble insulin, and 10 mEq potassium. The exercise studies were then repeated.

Both at rest and during exercise there was a conspicuous increase in left ventricular function in both groups of subjects. Cardiac output and left ventricular dp/dt (max) were conspicuously increased and left ventricular end-diastolic pressure substantially reduced at a similar heart rate and systemic arterial pressure to that in the control studies.

These findings indicate the potential therapeutic value of glucose in maintaining energy production in the ischaemic heart.

Delay times in acute ischaemic heart disease

J. M. Barber, D. McC. Boyle, *M. J. Walsh*, B. Shanks, and N. C. Chaturvedi (last three introduced)

The early mortality of myocardial infarction requires rapid intensive management. Mobile coronary care aims to reduce the total delay time between onset of symptoms and intensive care conditions. We admit patients via a mobile coronary care unit and conventionally.

We have studied: (1) The effect on patient delay time of age, sex, position of infarct, extent of infarct, severity of infarct as judged by a coronary prognostic index, previous ischaemic heart disease, cigarette smoking, diabetes mellitus, hypertension, time of initiation of in-

tensive care, and day of admission. (2) The patient's reasons for delaying (studied by means of questionnaire). (3) The impact of the mobile unit on delay times.

Our results suggest: (1) Patient delay time depends on time of day at which symptoms start and on the severity of the infarct. (2) Of patients delaying more than 30 minutes, 55 (56%) of 99 realized their pain was cardiac in origin. Of these, 21 (38%) considered early treatment to be important. (3) During the first two years total delay time shortened for mobile unit patients (due to reduction of doctor delay time). Patient delay time and mobile unit delay did not change. Delay times remained unaltered for conventional admissions.

Assessment of disordered left ventricular contraction from simultaneous measurements of left ventricular pressure and dimension

D. G. Gibson and David Brown (introduced)

Simultaneous measurements of left ventricular dimension by echocardiography and left ventricular pressure by catheter tip manometer were made in patients with valvular heart disease or cardiomyopathy. The time relations of movement in a localized part of the left ventricular cavity, defined by the echocardiographic dimension were thus compared with the overall ventricular response, reflected in the pressure. Stroke work and instantaneous and mean power were calculated and pressure dimension loops constructed for single cardiac cycles. In patients with normal ventricular function, the pressure dimension loop was approximately rectangular. The ratio of loop area to the product of maximum pressure and dimension changes in the cycle studied (cycle efficiency) was greater than 0·75. Left ventricular disease caused distortion of the loop and reduction in cycle efficiency due to loss of the optimal relation between pressure and dimension changes. Though peak power was increased, mean values were reduced by negative components in early or late systole. Cycle efficiency correlated closely with peak dp/dt, suggesting that disordered contraction is an important cause of impaired left ventricular function in valvular heart disease or cardiomyopathy.

Ventricular ischaemic response

A. F. Rickards, R. Wilkinson, W. Walsh (all introduced), and R. Balcon

The induction of cardiac pain by pacing is associated with a rise in left ventricular end-diastolic pressure.

The explanation of this phenomenon is obscure. Either the rise in filling pressure is associated with a concomitant increase in volume with no inherent change in the elastic properties of cardiac muscle, or the induction of angina is accompanied by local ischaemic changes which alter the pressure volume relation of the filling ventricle. Measurements of volume in the presence of possible regional changes are difficult to interpret.

We will present data from 50 patients who have undergone a pacing stress test with measurements of left ventricular pressure, and in some cases simultaneous right ventricular pressure and their derivatives, during pacing and after pacing interruption at successive heart rates. The findings will be correlated with the angiographic distribution of coronary artery disease and the presence or absence of angina in an attempt to elucidate whether the observed changes are regional or affect both ventricles.

Prevalence of lipoprotein abnormalities in the west of Scotland

A. R. Lorimer, V. M. Hawthorne, H. G. Morgan (both introduced), and T. D. V. Lawrie

One of the major risk factors in coronary heart disease is hyperlipidaemia. Previous studies have been mainly concerned with raised plasma cholesterol and/or triglyceride but newer concepts of disorders in lipid metabolism have led to interest in the relation of the hyperlipoproteinaemias to coronary heart disease. Studies of the lipoproteinaemias in population groups are few and so far have not been reported from the United Kingdom.

The prevalence of lipoprotein abnormalities was measured in 4474 apparently healthy male subjects. They were divided into 'normal' and 'abnormal' groups on the basis of blood pressure, electrocardiogram, obesity, cigarette smoking, and a history of possible previous angina or infarction. Classification of the hyperlipoproteinaemias was as suggested by Fredrickson, Levy, and Lees (1967).

The overall prevalence of type II hyperlipoproteinaemia was 3·3 per cent and of type IV was 12 per cent compared with 3·7 per cent and 7·2 per cent in the 'normal' and 3·1 per cent and 14·1 per cent in the 'abnormal' subgroups. Seventeen per cent of subjects who were obese, but otherwise normal, had a type IV abnormality, whereas the 3·5 per cent prevalence of type II abnormality was similar to that in other groups.

Thus in this region of Scotland lipoprotein abnormalities occurred in 15 per cent of an apparently healthy population. These results may be relevant to the high incidence of coronary heart disease in the area.

Reference

Fredrickson, D. S., Levy, R. I., and Lees, R. S. (1967). *New England Journal of Medicine*, **276**, 34.

Control of procainamide therapy after cardiac infarction

R. Royds, T. R. D. Shaw, *C. R. Kumana*, D. Padgham (all introduced), and John Hamer

Procainamide is an effective oral antiarrhythmic which is useful to prevent late recurrence of arrhythmias after cardiac infarction, but requires frequent doses to maintain adequate plasma concentrations.

We have assessed the effect of a standard regimen in

30 patients during the two weeks after the second or third day after cardiac infarction. Dosage was adjusted to maintain a plasma concentration between 4 and 8 μg/ml; this level is effective in suppressing ventricular arrhythmias without toxic effects.

Plasma levels were measured 1 and 4 hours after a 1 g loading dose. At 1 hour 5 patients had a level above 8 μg/ml but no toxicity was observed; 5 had a level below 4 μg/ml. Four hours after the loading dose, 17 patients had a level less than 4 μg/ml, and of these 5 were less than 3 μg/ml. The 5 patients with the lowest procainamide levels at 1 hour were atypical in that the 4-hour level was higher than the 1-hour level, indicating slow absorption. During maintenance treatment with 4-hourly doses related to body weight the levels showed considerable fluctuation.

Estimations of plasma procainamide levels are of great value in the control of treatment.

Measurement of regional myocardial blood flow in coronary artery disease

David Jewitt, B. Leonard Holman, Douglas F. Adams, Peter F. Cohn, James Adelstein, and Richard Gorlin (last five introduced)

Regional myocardial blood flow was measured in 25 patients with suspected coronary artery disease at rest and during rapid atrial pacing (15/25). Xenon was selectively injected into the left coronary artery and regional isotope distribution and washout monitored with an Anger scintillation camera. Scintiscans of isotope distribution in the myocardium were recorded and regional myocardial blood flow calculated over 4 quadrants in the left anterior oblique projection by the use of electronic cursors. Reproducibility of paired resting estimates was good ($R = 0{\cdot}97$, $P < 0{\cdot}001$).

In 9 patients with normal coronary vessels regional myocardial blood flow in the adjacent upper right and left and lower right and left quadrants was homogeneous at rest (i.e. $< 15\%$ difference). When 5 of these patients were atrially paced at rates in excess of 120/min regional myocardial blood flow in the upper quadrants rose by $19{\cdot}1 \pm 6{\cdot}1$ per cent and by $34{\cdot}8 \pm 4{\cdot}7$ per cent in the lower quadrants. Adjacent quadrants remained homogeneous on pacing.

Of 16 patients with coronary artery disease, 8 had isolated major occlusions in either the left anterior descending (6) or the left circumflex vessels (2) and regional myocardial blood flow was reduced in corresponding quadrants. In 2 further patients with left anterior descending occlusions though resting regional myocardial blood flow was homogeneous, pacing stress revealed impaired perfusion in the corresponding territory.

Regional myocardial blood flow determinations can show nonuniformity of perfusion characteristic of coronary artery disease and this nonuniformity, not always present at rest, can be unmasked by pacing stress.

Microangiopathic haemolytic anaemia in malignant phase hypertension

N. Oliver, H. Gavras, J. Aitchison, C. Begg, J. D. Briggs, J. J. Brown, P. W. Horton, F. Lee, A. F. Lever, C. Prentice, and J. I. S. Robertson (all introduced by J. D. Olav Kerr)

Microangiopathic haemolytic anaemia sometimes occurs in malignant phase hypertension. If the underlying disturbances of intravascular coagulation and haemolysis have a role in precipitating the malignant phase (see Gavras *et al.*, 1971), haematological abnormalities should be apparent in patients with malignant hypertension before the development of overt microangiopathic haemolytic anaemia.

This was tested by comparing haemoglobin, reticulocyte count, erythrocyte fragmentation, platelet count, serum fibrin degradation products, thrombin clotting time, kaolin-cephalin clotting time, urokinase sensitivity, factors V and VIII, euglobulin lysis time, one-stage prothrombin time, partial thromboplastin, and plasma plasminogen and fibrinogen in three groups of patients – group 1 comprised 10 patients with benign phase hypertension, group 2 comprised 11 patients with malignant hypertension but without overt microangiopathic haemolytic anaemia (Hb less than 65%, reticulocytes greater than 4%, and grade 2 erythrocyte fragmentation), and group 3 comprised 6 patients with malignant hypertension and overt microangiopathic haemolytic anaemia.

Haematological tests were generally normal in benign hypertension. Significantly lower values of haemoglobin, and platelet count with significantly higher values of reticulocyte count, erythrocyte fragmentation, serum fibrin degradation products, and urokinase sensitivity were found in groups 2 and 3 as compared with group 1. These results indicate that patients with malignant phase hypertension have haematological disturbance suggestive of microangiopathic haemolytic anaemia regardless of whether the condition is overt.

Reference

Gavras, H., Brown, W. C. B., Brown, J. J., Lever, A. F., Linton, A. L., MacAdam, R. F., McNicol, G. P., Robertson, J. I. S., and Wardrop, C. (1971). *Circulation Research*, **28**, Suppl. 2, 127.

Multiple dipole electrocardiography

P. W. Macfarlane (introduced), A. R. Lorimer, R. H. Baxter (introduced), and T. D. V. Lawrie

A mathematical model of the heart has been used to estimate the electrical activity of 10 discrete areas of the ventricular myocardium. The total e.m.f. of each myocardial segment is represented by a resultant electrical dipole, acting in a fixed direction. In practice, unipolar leads are recorded from 126 sites on the thorax using an on line computing technique. The QRS waveforms are later analysed by a digital computer to solve 126 simultaneous equations from which the onset, duration, and strength of the 10 electrical dipoles can be determined

throughout ventricular depolarization. Isopotential maps for the QRS-T segment can also be produced.

The technique has been applied to normal subjects and other patients with myocardial infarction, myocardial ischaemia, or conduction defects. A further small group has been studied before and after coronary artery surgery.

Areas of reduced electrical activity due to myocardial necrosis can be readily determined and where 12-lead electrocardiographic changes in patients with ischaemic heart disease were equivocal, the multiple dipole strengths were sometimes reduced compared to normal. While the technique is, at present, time consuming, it offers a noninvasive method of studying cardiac electrical activity in considerable detail.

Reference

Young, B. D., and Lawrie, T. D. V. (1971). Multichannel ECG data processing by computer. Proceedings of IEEE Conference, Sheffield.

Electrical safety: fibrillation thresholds with 50 Hz leakage currents in man and animals

E. B. Raftery, H. Green, and I. Gregory (both introduced)

The use of intracardiac catheters and pacing wires exposes patients to the danger of accidental electrocution should a current flow as a result of faulty equipment. Others have suggested that a leakage current as small as 10 μAmp can produce ventricular fibrillation but little is known of the physiological effects of leakage currents.

Accurate observations have been made of the physiological effects of 50 Hz leakage currents from catheters placed in the right atrium and right ventricle as follows: (1) 20 patients prepared for cardiopulmonary bypass; (2) 20 normal anaesthetized dogs; (3) 10 anaesthetized dogs after experimental myocardial infarction at intervals up to 6 months.

Ventricular fibrillation could not be induced from the right atrium in any group by currents up to 3 mAmp. The smallest current to produce ventricular fibrillation from the right ventricle was 80 μAmp in both man and animals. This threshold was not materially altered by myocardial infarction, but increased sensitivity was observed at two weeks post-infarction.

These observations have provided accurate standards for safety in the construction and maintenance of monitoring equipment.

Effect of verapamil on ventricular contraction

Winifred G. Nayler (introduced by Peter Harris)

Verapamil (5-(3,4 dimethoxyphenethyl) methylamino-2-(3,4-dimethoxyphenyl)-2-isopropylvaleronitrile) is an antiarrhythmic agent devoid of cardiac beta-adrenoceptor blocking activity. At dose levels of 0·3 and 0·5 μg/ml it reduced the peak tension (Tp) developed by isolated isometrically contracting dog trabecular muscles from control levels of $6{\cdot}01 \pm 1{\cdot}21$ and $6{\cdot}69 \pm 1{\cdot}39$ to $4{\cdot}10 \pm 0{\cdot}22$ ($P < 0{\cdot}02$) and $2{\cdot}16 \pm 0{\cdot}55$ ($P < 0{\cdot}002$) g/mm^2, respectively. Human heart muscle displayed a similar negative inotropic response. The reduction in Tp was accompanied by a significant ($P < 0{\cdot}02$) increase in the time taken to develop Tp. These negatively inotropic doses of verapamil failed to cause any significant change in the rate at which the sarcoplasmic reticulum (isolated as the microsomal fraction) either accumulated or bound Ca^{++}, but reduced the rate at which isolated but intact heart muscle accumulated Ca^{++}, as $^{45}Ca^{++}$. The negative inotropic effect of verapamil, even at relatively large (450 μg/ml) dose levels, is abolished under conditions that render the plasma membranes freely permeable to Ca^{++}. It is concluded that the negative inotropic effect of verapamil is due to a cell membrane located site of action which results in a reduced inwards displacement of Ca^{++} during excitation.

Noninvasive techniques for estimating isovolumic contraction time of left ventricle

T. Hardarson, *G. Ziady*, and R. Curiel (all introduced by J. F. Goodwin)

An important approach to the quantification of the contractile state of the left ventricle has been the study of the rate at which intraventricular pressure rises, or the first derivative of ventricular pressure (dp/dt). As the main determinant of the isovolumic contraction time is the dp/dt, hence the importance of estimating this time accurately.

Three methods for estimating the isovolumic contraction time were compared.

1) External isovolumic contraction time (EICT) derived from the carotid pulse tracing and phonocardiogram (S_1 A_2 – LVET).[1]
2) Pre-ejection period (PEP) calculated from the carotid tracing, phonocardiogram, and electrocardiogram (QA_2 – LVET).
3) Ultrasonic isovolumic contraction time (UICT) derived from the carotid pulse tracing, phonocardiogram, and mitral echogram, calculated as the distance from the B-point of the echogram to A_2, less LVET.

These recordings were performed in 10 normal subjects and 15 patients with hypertrophic obstructive cardiomyopathy, congestive cardiomyopathy, and ischaemic heart disease. In 11 patients the results were correlated with corresponding direct values (IICT, PEPI)[2] from intracardiac pressure curves. The UICT showed an excellent correlation with IICT (R: 0·92). The EICT correlated less well with IICT (R: 0·75) and was significantly shorter ($P < 0{\cdot}01$). The UICT and PEP had a superior discriminating value to EICT for differentiating the normals from the patients with ischaemic heart

[1] LVET: left ventricular ejection time.
[2] IICT: internal isovolumic contraction time.
PEPI: pre-ejection period internal.

disease and congestive cardiomyopathy ($P < 0.003$ and $P < 0.0005$ vs. $P < 0.07$).

The UICT is superior to the PEP in not containing components unrelated to the mechanical contractile process. Though more difficult to record than the EICT, the UICT is a convenient and reliable parameter of cardiac function.

Study of clinical pharmacology of hypertrophic obstructive cardiomyopathy by noninvasive diagnostic investigations

T. Hardarson and R. Curiel (both introduced by J. F. Goodwin)

Systolic time intervals were measured in 77 patients with hypertrophic obstructive cardiomyopathy, 16 of whom were studied during cardiac catheterization and 12 within 24 hours of catheterization. Control groups of 24 normal subjects and 129 patients with ischaemic heart disease and congestive cardiomyopathy were also studied.

The mitral echogram was used to supplement the phonocardiographic methods in determining the onset of mechanical systole.

The left ventricular ejection time index was found to be abnormally long in hypertrophic obstructive cardiomyopathy and highly dependent on the outflow tract gradient. This interval also correlated with the intensity of the systolic murmur, but not with the degree of mitral regurgitation.

In 7 patients with hypertrophic obstructive cardiomyopathy the mean left ventricular ejection time index was shortened from 435 to 410 msec after the intravenous administration of 0·5 mg phenylephrine, whereas in 4 control patients this interval was prolonged from 378 to 395 msec ($P < 0.001$). In 15 patients with hypertrophic obstructive cardiomyopathy the left ventricular ejection time index was prolonged from 409 to 422 msec after receiving 2 μg isoprenaline intravenously, while a shortening of the index (373 to 353 msec) was observed in 5 control patients ($P < 0.001$). The long-term oral administration of propranolol to 15 patients with hypertrophic obstructive cardiomyopathy was associated with a significant shortening of the isovolumic relaxation period, a decrease in the amplitude of the a-wave of the apex cardiogram, and a prolongation of the left ventricular ejection time index, all of which may be explained by improved ventricular distensibility.

The long left ventricular ejection time index observed in many patients and the paradoxical responses to phenylephrine and isoprenaline may be useful features in the diagnosis of hypertrophic obstructive cardiomyopathy.

Comparison of two cardioselective beta-adrenergic blocking agents: effects on haemodynamic and atrioventricular conduction in man

Marion Crouchman (introduced by John Hamer)

The effects of two cardioselective beta-adrenergic receptor blocking agents, acebutolol (M & B 17803a) and ICI 66,082, on cardiac output and atrioventricular conduction were compared at three equal accumulative intravenous dose levels (5, 12, and 30 mg).

Cardiac output was measured by the Fick principle at cardiac catheterization. All 14 patients in this group had mitral valve disease with atrial fibrillation. Both drugs produced a significant, dose-related fall in cardiac output and a reduction in oxygen consumption. There was no significant change in pulmonary vascular resistance, or arterial, indirect left atrial, or pulmonary arterial pressures.

The His bundle electrogram was recorded in 8 patients in sinus rhythm before and after administration of the beta-blocker, at rest and with atrial pacing at 4 increasing rates to compare the effects of the drugs independent of changes in heart rate. Both acebutolol and ICI 66,082 produced progressive prolongation of the AH interval with increasing doses.

Acebutolol (M & B 17803a) possesses intrinsic sympathomimetic activity, but ICI 66,082 does not. Some evidence of the sympathomimetic action of acebutolol was evident, but is not thought to be of clinical importance. Considerable reduction in cardiac output was produced in these patients by both drugs, though only acebutolol has quinidine-like activity.

Four years experience with Devices pacemakers

E. T. L. Davies, P. T. G. Hanson (both introduced), and H. F. M. Bassett

192 TF 2970 asynchronous and 80 demand pacemakers manufactured by Devices Ltd were implanted during the 4 years 1969–72. The ultimate fate of these units has been analysed.

Ninety-nine per cent of asynchronous and 89 per cent of demand units implanted for 12 months continued to function satisfactorily. Corresponding figures after 24 months were 77 and 70 per cent respectively. Units were electively replaced at 30 months, on the manufacturers' advice. Thirty units failed, the most frequent cause being leakage of electrolyte from a battery cell to the connexion pins, causing a low resistance path and premature discharge of the batteries. Sixteen units failed suddenly.

Implant lifetime may be curtailed for reasons other than pacemaker failure. The causes and frequency have been determined. The 272 units were implanted on 293 occasions, 21 units being used a second time after reconditioning. Seventy per cent of asynchronous and 69 per cent of demand units remained *in situ* after 12 months, 38 per cent and 44 per cent, respectively, after 24 months. Death from unrelated cause was the most frequent reason for curtailed pacemaker lifetime. Few data of this kind are available for comparison but these pacemaker lifetimes are similar to those reported from Glasgow (Green *et al.*, 1972) for a 4-year period (1966–70) using Medtronic units.

Reference

Green, G. D., Forbes, W., Shaw, G. B., and Kenmure, A. C. F. (1972). *American Heart Journal*, **83**, 265.

Developmental explanation for absence of coarctation in conditions with reduced pulmonary flow from birth

E. A. Shinebourne and A. M. Alseed (introduced)

Of 240 patients with coarctation of the aorta, none had an associated congenital cardiac abnormality resulting in diminished pulmonary blood flow. Review of the published material confirms the absence of coarctation in patients with tetralogy of Fallot. In the newborn, great vessel diameter reflects foetal flow patterns during late gestation. At birth the aortic isthmus (receiving ~25% of combined ventricular output) is normally 25 per cent narrower than the descending aorta (receiving ~60% of foetal flow). A shelf-like indentation of the posterior aortic wall opposite the ductus characterizes the junction of isthmus with descending aorta. In tetralogy of Fallot pulmonary atresia and tricuspid atresia, when pulmonary flow is reduced from birth, the main pulmonary artery is decreased and ascending aorta increased in size. Diminished main pulmonary artery flow reduces right-to-left shunting through the foetal ductus. The major proportion of descending aortic flow traverses the isthmus which is now the same diameter as the descending aorta. In pulmonary atresia pulmonary flow may derive entirely from the ductus, the isthmus is wider than the descending aorta, and intrauterine ductal flow is from left to right. The hypothesis presented is that foetal flow patterns preclude development of isthmal narrowing or juxtaductal coarctation when main pulmonary artery flow is reduced in the foetus.

Left ventricular aneurysm: current appraisal

Magdi Yacoub, E. Knight (introduced), Malcolm Towers, and Walter Somerville

In this paper an attempt is made to define accurately what constitutes a left ventricular aneurysm, as we have found that occasionally what looks like a left ventricular aneurysm angiographically, may be found at operation to be an area of partially scarred or viable but poorly perfused myocardium which moves paradoxically. Accordingly, various criteria for the definition of aneurysm have been evaluated.

Between October 1969 and January 1973, 38 patients with chronic left ventricular aneurysm were treated surgically. There were 34 men and 4 women ranging in age between 36 to 70 years. All patients had angina, left ventricular failure, or both. Haemodynamic and angiographic studies were performed in all patients. Complete occlusion of one major coronary vessel or severe narrowing of 2 or 3 important vessels was present in all patients. The diagnosis was suspected clinically, before angiography, in the majority of patients.

At operation, the ventricular septum was scarred in all patients and 1 patient had a postinfarction ventricular septal defect which was repaired at the same time. Scarring of one or both papillary muscles was present in 6 patients, but significant mitral regurgitation requiring valve replacement was present in only 1. Coronary artery bypass graft was performed at the same time in 18 patients. There were 2 operative deaths (5%) and 2 late deaths. The degree of clinical improvement correlated well with the state of the remaining myocardium as judged by preoperative angiography and during operation.

It is concluded that the preoperative diagnosis of left ventricular aneurysm should depend on rigid angiographic criteria. When symptomatic, surgical excision with or without myocardial revascularization can result in conspicuous improvement in clinical state.

Primary surgical correction of total anomalous pulmonary venous drainage in infants

Rosemary Radley-Smith and Magdi Yacoub

Between October 1970 and December 1972, 6 infants ranging in age between 9 days and 2 years (5 were below the age of 5 months) underwent primary repair of total anomalous pulmonary venous drainage at Harefield Hospital. The operation was performed as an emergency in 5 because of rapidly deteriorating clinical condition.

The pulmonary venous drainage was supracardiac in 5 and intracardiac in 1. Obstruction of the common pulmonary venous trunk was present in 1.

Surface induced profound hypothermia with rapid rewarming using cardiopulmonary bypass was used in all except 2 early in the experience, in whom normothermic bypass and core cooling respectively were used. For supracardiac total anomalous pulmonary venous drainage a large open anastomosis between the left atrium and common pulmonary venous channel, using a new technique, was used. For intracardiac total anomalous pulmonary venous drainage excision of the interatrial septum and insertion of a pericardial baffle was used. There were 2 postoperative deaths, 1 before adopting the current technique of surface induced profound hypothermia and 1 due to renal failure 10 days postoperatively in a 9-day-old infant with obstructed total anomalous pulmonary venous drainage who was *in extremis* on admission.

It is concluded that primary repair in infancy is the method of choice for treating patients with total anomalous pulmonary venous drainage. There is no place for palliative septostomy.

Spectrum of floppy mitral valve syndromes: clinicopathological study

Magdi Yacoub and *Roxane McKay* (introduced)

Myxomatous degeneration of the mitral valve cusps and chordae results in mitral regurgitation due to prolapse of

one or both cusps. Depending on the site and severity of the lesion, several clinicopathological syndromes result and probably represent different stages in the natural history of the disease. Between September 1969 and January 1973, 47 patients with floppy mitral valves were seen at Harefield Hospital, excluding those with papillary muscle dysfunction due to coronary artery disease or cardiomyopathy. In 44 patients (26 men and 19 women) the lesion was severe enough to require operative correction. An asymptomatic heart murmur in 24 patients preceded the onset of symptoms by as long as 65 years. These were of 3 types: a mid or late systolic murmur which often progressed to pansystolic; a systolic ejection murmur radiating to the base (invariably associated with ruptured chordae to the posterior cusp) and a pansystolic murmur radiating to the axillae. At the time of operation all patients were judged to be in class III or IV of the New York Heart Association Classification. Once symptomatic, all patients suffered progressive haemodynamic decompensation, the period between onset of symptoms and operation being less than 18 months in the majority. One patient presented with acute mitral regurgitation, due to rupture of multiple chordae, and required emergency operation because of persistent cardiogenic shock. In all cases the cardiac index was low, ranging from 1·2 to 2·4 l./m^2 per min (mean = 1·6 ± 0·36). A family history of valvular disease was present in 3 cases, skeletal stigmata of the Marfan syndrome in 3, and past history of infective endocarditis in 7.

Detailed histological examination showed diffuse myxomatous change throughout both cusps as well as circumferential deposition of myxomatous tissue around chordae tendineae and disorganization of the collagen core. Aortic biopsies often revealed medial degeneration.

It is concluded that myxomatous change can result in mitral regurgitation of a significant degree and may be associated with more diffuse abnormalities in the cardiovascular system. Though patients may remain in a compensated state for many years, once cardiac failure starts rapid progression usually occurs.

Tape monitoring of electrocardiogram in ambulant patients with sinoatrial disease

B. R. M. Crook, P. M. M. Cashman, R. D. Stott (all introduced), and E. B. Raftery

Intermittent slow and fast dysrhythmias are a recognized feature of sinoatrial disease, but little is known about the range and frequency of these rhythm disorders in the individual patients. Continuous electrocardiogram recordings have been taken over a 3-day period in 25 fully ambulant patients using a miniature tape-recorder with 24-hour cassettes. The recordings were analysed using specially developed automatic dysrhythmia detection, based on RR interval discrimination. The sampled dysrhythmias were written out as a conventional electrocardiographic trace. Twelve-lead electrocardiographic records on these patients had shown episodes of sinus bradycardia (rate less than 56/minute) in 76 per cent, 2nd-degree sinoatrial block in 44 per cent, 3rd-degree sinoatrial block in 16 per cent, atrial fibrillation in 4 per cent, and no episodes of supraventricular tachycardia or sinus tachycardia (rate greater than 110/min). With the additional information from the tape recordings, the incidence of dysrhythmias detected increased to: sinus bradycardia in 88 per cent, 2nd-degree sinoatrial block in 64 per cent, 3rd-degree sinoatrial block in 36 per cent, atrial fibrillation in 32 per cent, supraventricular tachycardia in 52 per cent, and sinus tachycardia in 44 per cent. This high incidence of different dysrhythmias and especially of brief episodes of tachydysrhythmias detected from the tape records has confirmed the wide spectrum of intermittent rhythm disorders in these patients with sinoatrial disease. The technique of tape monitoring, by showing the characteristic dysrhythmias and their severity, is valuable for the diagnosis and treatment of this disorder.

Progression of coronary artery disease: a clinical arteriographic study

Richard Gorlin and Charles E. Bemis (both introduced by J. F. Goodwin)

Significant progression of coronary artery disease was seen in 52 per cent of subjects studied by selective cinearteriography at intervals between 2 and 75 months (average 23·8). Subsequent progression, though confined to proximal areas, was independent of overall severity of initial disease or previous disease at the site of progression and occurred frequently in previously normal vessels. Plasma lipid abnormalities and myocardial lactate production at the time of the initial study were significantly associated with subsequent arteriographic progression. Similarly abnormal glucose tolerance was seen more frequently in patients exhibiting progression than in those who did not. The progression occurring in patients with lipid abnormalities was more severe and more widespread than in other patients and apparent interval reduction in lipid values did not influence the ultimate course of the atheromatous process. Myocardial infarction was almost invariably associated with progression. Collateral coronary circulation never increased or appeared unless accompanied by an increase in the extent of local coronary artery disease. The absence of progression was associated with a favourable prognosis. All other clinical, laboratory, and arteriographic parameters analysed were not predictive of subsequent progression of the coronary obstructive lesion.